

Remarks

Amendments to the Claims

The amendments to the claims do not add new matter. Independent claim 2 is amended to recite a step of determining whether the test compound has an effect on a symptom of heart failure in an *in vivo* assay. The specification supports this amendment on page 39, lines 10-16. Claim 2 also is amended to recite that the activity of the FPRL2 is an activity of a G protein coupled receptor. New claim 37 recites that the activity is measured by an alteration in intracellular calcium concentration. The specification supports new claim 37 and the amendment to claim 2 on page 41, lines 4-14.

Rejections Under 35 U.S.C. § 101 and § 112 ¶ 1

The Office Action maintains the rejection of claims 2, 27, 28, and 32 under 35 U.S.C. § 101 as lacking utility, with a corresponding rejection for lack of enablement under 35 U.S.C. § 112 ¶ 1. Applicants respectfully traverse the rejections.

The Office Action makes several assertions of why Applicants' previous response was not persuasive. None of these assertions supports the utility and enablement rejections.

First, on page 5 the Office Action asserts that identification of potential therapeutic compounds for treating heart failure is not a specific and substantial utility because the specification also discloses other disorders which may be addressed using regulators of the recited FPRL2 protein. A "specific" utility does not mean a "unique" utility; although the specification needs to assert only one credible assertion of specific utility, *Raytheon v. Roper*, 724 F.2d 951,958, 220 U.S.P.Q. 592, 598 (Fed. Cir. 1983), *cert. denied*, 469 U.S. 835 (1984), this does not preclude the specification from asserting more than one utility. Citing to page 55 of

the specification, the Office Action also contends that heart failure “comprises many different pathological states with distinct pathological features (page 55).” *Id.* Page 55 of the specification defines “heart failure” as:

a pathophysiological state in which an abnormality of cardiac function is responsible for the failure of the heart to pump blood at a rate commensurate with the requirement of the metabolizing tissue. It includes all forms of pumping failures such as high-output and low-output, acute and chronic, right-sided or left-sided, systolic or diastolic, independent of the underlying cause.

Page 55, lines 8-12. The common pathological feature of these states, however, is failure of the heart to pump blood at an appropriate rate. Nothing in this definition suggests that a screening method for a test compound which affects a molecule involved in contraction/relaxation (see below) would not be useful for identifying potential therapeutic agents for treating heart failure.

Second, in the paragraph bridging pages 5 and 6, the Office Action points to the relative expression of FPRL2 mRNA in the whole heart as being “quite low” and faults Table 1 for not showing “predominant expression of FPRL2 polypeptide in heart or in cardiomyocytes.” The Table 1 discloses high expression of FPRL2 mRNA in the right atrium and ventricle, whereas the expression of FPRL2 in the whole heart is an average of the expression in various parts of the heart. As Applicants explained in the response filed May 9, 2008, those of skill in the art know that the right atrium and ventricle contain cardiomyocytes.

Third, on page 6 the Office Action asserts that neither the prior art nor the specification “establishes that the FPRL2 polypeptide modulates myocardial contractility.” Keitoku,¹ which is of record, discloses a contraction/relaxation effect of FMLP, a ligand which binds to the three FMLP receptors, including FPRL2, in coronary arteries. This publication supports use of FRPL2

¹ Keitoku *et al.*, “FMLP Actions and its Binding Sites in Isolated Human Coronary Arteries,” *J. Mol. Cell. Cardiol.* 29, 881-94, 1997; of record in the IDS filed September 22, 2004.

as a target for treating heart failure which, as explained above, involves a failure of an appropriate rate of pumping (*i.e.*, contraction of cardiomyocytes).

Finally, on page 6 the Office Action insists that a causative link is required between FPRL2 polypeptide and a specific type of heart failure in order for one skilled in the art to know whether an agonist or antagonist of FPRL2 is useful for treating heart failure. This knowledge is not required for the claimed screening method to be useful. As explained in the response filed May 9, 20008, heart failure occurs because heart muscle is damaged or dead. Current treatments for heart failure do not restore the damaged muscle or cause heart cell regeneration. Thus, a causative link to a heart failure is not required for a particular drug target to be useful for treating heart failure.

The Office Action does not establish a *prima facie* case that the claimed screening method is not useful or enabled. Please withdraw the rejections.

Rejections Under 35 U.S.C. § 112 ¶ 2

Claims 2, 27, 28, and 32-36 are rejected under 35 U.S.C. § 112 ¶ 2 as indefinite. The Office Action contends that independent claim 2's recitation of "the activity of a formyl peptide receptor-like 2" is indefinite. To advance prosecution, claim 2 is amended to recite that the activity of the FPRL2 is an activity of a G protein coupled receptor. Please withdraw the rejection.

Objection to Claim 27

Claim 27 is objected to because it depends from a canceled claim. Claim 27 is amended to depend from pending claim 32. Please withdraw the objection.

Respectfully submitted,

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